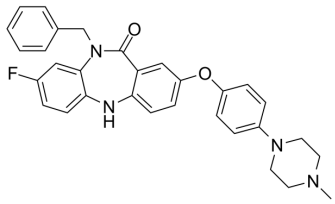
Mutated EGFR-IN-3

Cat. No.:	HY-130608
CAS No.:	2375107-27-8
Molecular Formula:	$C_{31}H_{29}FN_4O_2$
Molecular Weight:	508.59
Target:	EGFR
Pathway:	JAK/STAT Signaling; Protein Tyrosine Kinase/RTK
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Mutated EGFR-IN-3 (compound 3) is a potent, ATP-competitive and highly selective allosteric dibenzodiazepinone inhibitor of the EGFR(L858R/T790M) and EGFR(L858R/T790M/C797S) mutants with IC_{50} values of 12 nM and 13 nM, respectively[1].	
IC_{50} & Target	$EGFR^{L858R/T790M}$ 12 nM (IC_{50})	$EGFR^{C797S}$ 13 nM (IC_{50})
In Vitro	Mutated EGFR-IN-3 inhibits EGFR through an allosteric mechanism, biochemical IC_{50} values at varying ATP concentrations: 10 μM, 10.0 μM, 100.0 μM, 1000.0 μM are 15 nM, 8.3nM,11.0 nM and 8.3 nM, respectively for L858R/T790M cells[1]. Mutated EGFR-IN-3 exhibits antiproliferative activities of a panel of EGFR allosteric inhibitors are 7.0 μM, 3.3 μM, 3.8 μM, 4.0 μM and 4.5 μM for parental, WT, L858R, L858R/T790M and L858R/T790M/C797S, respectively in the absence of Cetuximab in Ba/F3 cells[1]. Mutated EGFR-IN-3 exhibits antiproliferative activities of a panel of EGFR allosteric inhibitors are 3.2 μM, 2.7 μM, 0.36 μM and 0.20 μM for WT,L858R, L858R/T790M and L858R/T790M/C797S, respectively in the presence of Cetuximab in Ba/F3 cells[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.	

REFERENCES

[1]. De Clercq DJH☒et al. Discovery and Optimization of Dibenzodiazepinones as Allosteric Mutant-Selective EGFR Inhibitors.ACS Med Chem Lett. 2019 Oct 22;10(11):1549-1553.

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